# Maternal Depression and Stress Response

## The Effect on Offspring in Emerging Adulthood

by

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ABSTRACT

Dysregulated cortisol has been linked to a variety of adverse physical and psychological consequences. Stressors in the childhood family environment can influence cortisol activity throughout development. For example, research has shown that both infants and children of depressed mothers exhibit altered levels of cortisol compared to infants and children of non-depressed mothers. It is unclear, however, whether exposure to maternal depression in childhood and adolescence is related to cortisol activity at later stages of development. The current study examined the longitudinal relation between maternal depressive symptoms during late childhood (9-12 years old) and adolescence (15-19 years old) and cortisol activity in offspring in young adulthood (24- 28 years old) in a sample of 40 young adults and their mothers. Maternal depressive symptoms were prospectively assessed at four time points across the 15 year study. Cortisol samples were collected from young adult offspring at the final time point. Findings revealed that higher levels of maternal depressive symptoms during late childhood were associated with lower total cortisol output in young adulthood. Results suggest that attenuated cortisol levels, which put these young adults at risk for a variety of stress-related physical and psychological illnesses, may be a long-term consequence of exposure to maternal depression,. Depressive symptoms in mothers during their child's adolescence, however, did not relate to cortisol output. These findings suggest a sensitive period in late childhood during which the development of HPA activity may be susceptible to the environmental stressor of maternal depression.

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# INTRODUCTION

Depression affects approximately one in five women (APA, 2000), a lifetime prevalence rate greater than any other psychiatric disorder affecting women in the United States (Kessler et al., 1994). Mothers who have recently experienced divorce are at particular risk of developing depression (Lorenz, Wickrama, Conger, & Elder, 2006). Divorce is often characterized by a variety of stressful life changes including economic hardship (Amato, 1993; Fischer, 2007), role changes (Martinez & Forgatch, 2002), and increased family conflict (Buchanan, Maccoby, & Dornbusch, 1991) that may contribute to the heightened risk for maternal depression. Depression in women is often accompanied by many deleterious effects, and the consequences are amplified when the woman with depression is raising children.

Maternal depression has been linked to a variety of mental health consequences for offspring. For example, children of depressed mothers are at elevated risk for both internalizing and externalizing mental health problems (Ashman, Dawson, & Pantigiotides, 2008; Goodman & Gotlib, 1999; Martins & Gaffan, 2000; Murray, Fiori-Cowley, Hooper, & Cooper, 1996; Weissman, Warner, Wickramaratne, Moreau, & Olfson, 1997). Evidence strongly suggests that these mental health consequences can endure into adulthood. For example, offspring of women who experienced depression are more likely to develop depression themselves in adulthood and the depression is likely to have an earlier onset and greater severity (Ensminger, Hanson, Riley, & Juon 2003; Lieb, Isensee, Hofler, Pfister, & Wittchen, 2002). Young adults with mothers who were

depressed during their childhoods are also at increased risk for more severe anxiety disorders (Rohde, Lewinsohn, Klein, & Seeley, 2005). Further, maternal depression puts adult offspring at increased risk for alcohol dependence (Weissman et al., 1997).

In addition to mental health consequences, maternal depression is linked to adverse physical health outcomes in children including dysregulation of stress response systems (Ashman, Dawson, Pantigiotides, Yamada, & Wilkinson, 2002; Essex, Klein, Cho, & Kalin, 2002; Gump et al., 2009; Murray, Halligan, Goodyer, & Herbert, 2009). A dysregulated stress response is a risk marker for the development of both psychological and physical health problems including depression, anxiety, externalizing disorders, and cardiovascular disease (McEwen, 2000; McEwen, 1998). To date, studies have not examined the relation between maternal depression and stress response in young adults so it unclear whether maternal depression in childhood has enduring physical health consequences on children as they age. Examination of this research issue is important because a dysregulated stress response system in adulthood is associated with serious mental and physical health consequences.

Stress response is often measured by cortisol, the end product of the hypothalamic pituitary adrenal (HPA) axis response to stress. The HPA axis functions as an adaptive response system to maintain optimal functioning in a stressful environment. In the face of a stressor, the HPA axis releases cortisol to help the individual cope with the stressor (Nicolson, 2008). The release of cortisol leads to increased blood glucose levels, cardiovascular activity, and alertness,

changed cognitive and sensory thresholds, and suppression of nonessential functions (e.g., growth, digestion), which increase the amount of energy available to deal with the stressor (Davies, Sturge-Apple, Cicchetti, & Cummings, 2007).

The HPA-axis is highly susceptible to chronic environmental stressors. Early life stress experiences, in particular, can lead to cortisol dysregulation through psychosocial, and/or cognitive/affective pathways (Luecken & Lemery, 2004). Cortisol dysregulation can take two forms: hypercortisol and hypocortisol. Historically, studies identified increased cortisol, or hypercortisol, following exposure to early life stress (e.g., Breier et al., 1988; Gunnar & Vazquez, 2001; Luecken, 2000). More recently, studies have emerged reporting that deficient production of cortisol, or hypocortisol, also can result from early life stress (for a review see Heim, Ehlert, & Helhamer, 2000). Both hypercortisol and hypocortisol have been linked to adverse physical and psychological consequences (Cohen, Kessler, & Underwood, 1995). It remains unclear, however, what conditions lead to increased versus decreased cortisol levels. Miller, Chen, and Zhou (2007) propose that the timing of the stressor determines the direction of the effect on the HPA-axis. The more immediate effect of an environmental stressor is hypothesized to be hypercortisol, whereas the longer-term effect of a stressor is thought to be hypocortisol.

Evidence shows that maternal depression is an early life stressor that is associated with changes in children's cortisol activity. Several studies that have examined the relation between postpartum depression and cortisol in infants have consistently found elevated cortisol levels in offspring of depressed mothers

relative to those of non-depressed mothers (e.g., Dougherty, Klein, Olino, Dyson, & Rose, 2009; Essex et al., 2002). Only a handful of studies have examined the relation between maternal depression and cortisol in offspring later in development, and the results of these studies have been less consistent than the findings for infants. Examining 7 and 8 year olds, Ashman and colleagues (2002) showed that children with high internalizing problems who had mothers with a history of depression showed higher cortisol levels than children of non-depressed mothers. In this study, maternal depression in the first two years of the child's life was the best predictor of elevated cortisol. Studying adolescents, Murray et al. (2009) showed that maternal withdrawal in postnatal depressed mothers predicted higher cortisol awakening response. In a sample of offspring from 17 to 21, Mannie, Harmer, and Cowen (2007) found that participants with a history of family depression had a higher cortisol awakening response than participants without a family history of depression.

However, other studies have reported either attenuated levels of cortisol among offspring of depressed mothers relative to those whose mothers were not depressed or a non-significant relation between maternal depression and cortisol response. For example, Fernald, Burke, and Gunnar (2008) studied children, ages 2.5 to 6, and found that higher levels of maternal depressive symptoms were related to lower levels of cortisol. Examining the relations between maternal depressive symptoms and stress response in 10 year old children during a stress response task, Gump and colleagues (2009) showed that chronic maternal depression was related to significantly lower levels of cortisol. Contrary to earlier

findings, maternal depression when the child was an infant did not predict abnormal cortisol levels and neither early nor chronic depression was related to cortisol reactivity. Ronsaville and colleagues (2006) compared cortisol reactivity in adolescents with healthy mothers to adolescents whose mothers had a past diagnosis of unipolar depression or bipolar disorder using a corticotrophin-releasing hormone (CRH) challenge test. They found no main effects of maternal psychopathology, but found that family stress was negatively associated with offspring cortisol reactivity.

Reasons for these discrepant findings are not readily apparent; however, the timing of maternal depression may be an important factor in the relation between depression and cortisol response. Most studies show that exposure to maternal depression very early in the child's life is associated with hypercortisol levels (Ashman et al., 2002; Dougherty et al., 2009; Essex et al., 2002; Murray et al., 2009) whereas maternal depression later in the offspring's development has not been consistently associated with an elevated response (e.g., Fernald et al., 2008). Additional research is needed to examine whether the relations between maternal depression and cortisol response differ depending on the timing of maternal depression.

There are several possible mechanisms that might explain the adverse effects of maternal depression on offspring's stress response. First, maternal depression may negatively affect parenting behavior. For example, maternal depression has been linked to a variety of suboptimal parenting strategies including inconsistent discipline, increased negative affect, decreased positive

affect, and disengagement from the child (Goodman & Gotlib, 1999; Lovejoy et al., 2000). Further, evidence has shown significant associations between adverse parenting experiences and dysregulated HPA stress-responses (DeBellis, 2001; Bugental, Martorell, & Barraza, 2003). Thus, parenting may be one mechanism explaining the relation between maternal depression and offspring stress response.

Alternatively, mothers with depression might model maladaptive coping and emotion regulation strategies that contribute to the development of a dysregulated stress response. Depression has been linked to impaired emotion regulation strategies in mothers, characterized by increased negativity, hostility, and ineffective problem solving (Blandon et al., 2008; Goodman & Gotlib, 2002; Gump et al., 2009). Researchers have suggested that depressed mothers might model maladaptive coping strategies or have difficulty coaching their children to use adaptive emotion regulation responses (Gross & Munoz, 1995; Silk, Shaw, Skuban, Oland, & Kovacs, 2006). Poor emotion regulation strategies have been shown to relate to a dysregulated stress response (Repetti et al., 2002), therefore parent modeling may explain the association between maternal depression and offspring cortisol activity.

The proposed study will use the control group in a longitudinal study of a preventive intervention for divorced mothers and children to examine the relations between maternal depressive symptoms and cortisol activity in young adult offspring. Specifically, the study will assess the relation between level of depressive symptoms in mothers when their offspring were in late childhood and adolescence and cortisol levels and cortisol reactivity in response to a stress task

in offspring in young adulthood. It is hypothesized that young adults whose mothers had higher levels of depressive symptoms during the offspring's late childhood and adolescence will exhibit lower cortisol levels and blunted cortisol reactivity compared to young adults exposed to less severe maternal depressive symptoms. Hypocortisol and a blunted stress response are hypothesized because maternal depression can be characterized as a long-term versus short-term stressor. Additionally, the study will examine the differential effects of patterns of maternal depressive symptoms during the offspring's late childhood and adolescence. The effect of the patterns of maternal depressive symptoms will be examined by assessing the unique contributions of depressive symptoms that occurred during offspring's late childhood, depressive symptoms that occurred during adolescence, and the interaction between the two time points. It is hypothesized that higher maternal depressive symptoms in offspring's late childhood, as well as exposure to maternal depressive symptoms in both late childhood and adolescence will be more detrimental than no maternal depressive symptoms or higher symptoms occurring only in adolescence.

This study will extend knowledge about the relation between exposure to maternal depression and cortisol activity in offspring by examining this relation at a later stage in development than has been examined in previous research. This extension of the literature is important because brain development continues through adolescence (Chugani, 1988), and the examination of the HPA stress-response system in young adulthood, when brain development is relatively stabilized, could provide a clearer picture of the long-term effects of maternal

depression on offspring's physiological functioning. Further, this study contributes to research in this area by its focus on the effects of patterns of the timing of the maternal depression on stress response in young adulthood. By exploring the differential influence of patterns of maternal depression, the findings may assist in the development of interventions that target depressed mothers.

## METHODS

### *Participants*

The control group from the New Beginnings Project (NBP), a randomized trial examining the efficacy of an intervention for divorced mothers and their children, was used (Wolchik et al., 2000). Families were randomly assigned to one of three conditions: a mother-only program, a separate mother and child program, or a literature control condition. Participants were identified primarily by using court records of randomly selected divorce cases in the Phoenix, Arizona metropolitan area. Twenty percent of the families were recruited via media advertisements. Eligible mothers had a child between the ages of 9 and 12 and had experienced a divorce within the past 2 years. Additionally, the mother was the primary caregiver, mother had no live-in boyfriend or plans to re-marry during the course of the NBP study, neither the mother nor child was in treatment for mental health problems, both mother and child were fluent in English, the child was not learning disabled or handicapped, and any child diagnosed with attention-deficit/hyperactivity disorder was taking medication. Exclusion criteria included

children who exhibited suicidality or severe levels of depression or externalizing behavior problems at pre-test. These families were referred for treatment.

Seventy-six families were randomly assigned to the control group. Data were collected at six time points: baseline (Time 1), post-test (Time 2), 3-month follow-up (Time 3), 6- month follow-up (Time 4), 6-year follow-up (Time 5), and 15-year follow-up (Time 6). The current study will use data from time points 1, 2, 5, and 6. In the control group, 76 families completed assessments at Time 1 and 2, 68 completed Time 5, and 60 completed Time 6. Sixty families completed all follow-up assessments, including the final time point. Cortisol data was gathered at Time 6. Of the 60 young adults (YAs) interviewed at Time 6, 2 refused to participate in the cortisol assessment, 1 lived out of the country and was not administered the cortisol assessment, and 1 was unable to complete the cortisol assessment due to scheduling conflicts.

Study variables were examined to identify participants with extreme scores. One YA's cortisol scores were an average of 4.8 standard deviations above the mean of the data. This case was removed from analyses. No other extreme scores were found. Additionally, the data were examined to see if any YAs should be eliminated because of the following health-related conditions that have been shown to be related to cortisol output and reactivity: medications (i.e., systemic glucocorticoids, anticonvulsants, hormone replacement medications, beta blockers, and steroids; Nicolson, 2007), medical conditions (i.e., Type I diabetes, endocrine disorders, adrenal disorders, autoimmune disorders; Nicolson, 2007) or pregnancy (Kudielka, Hellhammer, & Wst, 2009). Two YAs with

hypothyroidism and five YAs who were pregnant were removed from the analyses. Additionally, four YAs whose mothers did not have data at Time 5 and four YAs with incomplete cortisol data were eliminated from analyses. The sample used in analyses consisted of 40 YAs and their mothers.

T-tests and chi-squared tests were used to compare participants excluded from the analyses (n = 16) to participants included in the analyses (n = 40) on four Time 1 demographic variables (i.e., age, gender, ethnicity, income) and Time 1 maternal depression. No significant differences were found.

The mean age of the YA participants in the current study was 25.5 (SD = 1.2) years. At the first time point, the mean age of participants was 10.3 (SD = 1.1) years and the mean age of mothers was 36.3 (SD = 4.6) years. Fifty percent (n = 20) of the YAs were male; 85% Caucasian, 10% Hispanic, 2.5% Black, and 2.5% other. At Time 1, the median family income ranged from $20,000 to $25,000 and 80% of mothers had completed at least some college.

*Procedures*

Participants were assessed at six time points as described above. At all time points, nearly all participants completed the assessment in their home; the remainder completed the assessments at the research center. Trained staff conducted separate interviews with mothers and youth/young adults. Confidentiality was explained and parents and youth signed consent/assent forms. For the first five assessments, the interviewer verbally administered all questionnaires using a structured computer program. At Time 6, interviewers administered questionnaires to mothers via telephone. Research has shown data

gathered across different modalities (e.g., telephone vs. in-person survey) to be highly comparable (e.g., Aziz & Kenford, 2004). Families received $45 compensation at T1, T2, and T4; mothers and youth each received $100 compensation at T5; and mothers received $50 and young adults received $100 compensation at T6. All research procedures were reviewed and approved by the Institutional Review Board at Arizona State University.

*Measures* (See Appendix A for measures)

*Maternal Depressive Symptoms.* The Psychiatric Epidemiology Research Interview (PERI; Dohrenwend et al., 1980) was administered at time points 1, 2, 5, and 6. This 27-item self-report measure assesses general demoralization, or non-specific psychological distress, and consists of subscales measuring dread, anxiety, sadness, helplessness, hopelessness, psychophysiologic symptoms, perceived physical health, poor self-esteem, and confused thinking. Each item is rated on a 5-point response scale (0= *Never*, 1=*Almost Never*, 2=*Sometimes*, 3=*Fairly Often*, 4=*Very Often*). Participants with a mean score of 1.55 or greater are considered "demoralized" (Shrout,Dohrewend, & Levav, 1986). Dohrenwend et al. (1980) reported that all subscales have acceptable reliabilities (alpha coefficients; range = 0.80 to 0.85) and advised combining the subscales to form a summary score. The summary score has shown evidence of construct validity (Dohrenwend et al., 1980) and is useful for detecting cases of depression in community samples (Roberts & Vernon, 1981). Additionally, the latent structure of the PERI was compared to the Beck Depression Inventory and both were found

to load on the construct of depression (Tanaka & Huba, 1984). In the current study, the response format was adapted to ask about the past month.

Two scores were computed: maternal depressive symptoms in late childhood (MD-child) and maternal depressive symptoms in adolescence (MD-adol). MD-child (youth were 9 – 12 years old), was calculated by taking an average PERI score from Time 1 and 2. MD-adol (youth were 15 – 19 years old) was calculated using the PERI score from Time 5.

*Cortisol.* Cortisol samples were collected at Time 6 only. Samples were taken between 6:00pm and 10:00pm during an assessment consisting of two stressor tasks: mental arithmetic (Cacioppo et al., 1995) and a social stressor task (van Eck, Nicolson, Berkhof, & Sulon, 1996). The three-minute mental arithmetic task required YAs to perform serial subtraction aloud, starting from a new number each minute. The task was adjusted for difficulty and was timed to add more pressure. Immediately after the mental arithmetic task, participants completed the social stressor task. In this task, after 4 minutes of preparation, YAs gave a 4-minute videotaped speech about their strengths and weaknesses. Participants were told that a team of psychologists would evaluate the tapes. .Many studies have shown these tasks have significant effects on cortisol stress response (e.g., Cacioppo et al., 1995; see Dickerson & Kemeny's review, 2004).

YA participants were instructed to refrain from consumption of food, alcohol or caffeine two hours prior to the cortisol task. They were also asked not to brush their teeth or exercise two hours prior to the task. Saliva samples were collected at four periods: before the arithmetic task (P1), immediately after the

completion of the second task (P2), and again 20 minutes (P3) and 40 minutes later (P4). Saliva was collected with a small cotton roll held against the participant's inner cheek for 2 minutes using a Salivette (Sarstedt, Rommelsdorf, Germany) device. Saliva samples were then frozen at 20°C and mailed overnight to Salimetrics, Inc. where they were assayed for salivary cortisol using a high-sensitive enzyme immunoassay. This immunoassay test has a range of sensitivity from .007 to 1.8, and average intra- and inter-assay coefficients of variation of 4.13% and 8.89%, respectively. The test was designed by the Penn State University Behavioral Endocrinology Laboratory, and the assay's QA/QC has been independently verified by researchers at the UCLA Center for Health Science. Two measures of cortisol were analyzed: total output and cortisol reactivity to the stressor tasks.

*Potential covariates.* At Time 1, mothers completed a brief measure assessing demographic information including age, gender, education, and income. Young adults and mothers completed similar measures at Time 6.

Before the cortisol assessment, YAs completed a brief health survey including information on variables have been shown to relate to cortisol activity (Nicolson, 2007) (e.g., meals, exercise, smoking, and caffeine intake in the past 2 hours, and medication taken in the past 24 hours).

Internalizing and externalizing problems of YAs at Time 6 were measured using the Adult Self-Report (ASR; Achenbach, Dumenci, & Rescorla, 2003) and the Adult Behavior Checklist (ABC; Achenbach, Dumenci, & Rescorla, 2003). The ASR, completed by the YA, and the ABC, completed by the mother, are each

137-item measures developed for 18-59 year olds. Both have shown good internal consistency and test-retest reliability. A composite score of YA and mother report of internalizing and externalizing problems was created by taking the mean of the t-scores from each reporter. Internalizing and externalizing symptoms have been shown to be associated with cortisol activity (Scerbo & Kolko, 1994).

## RESULTS

### *Preliminary Analysis*

*Descriptive statistic summary:* Table 1 shows the descriptive statistics of the sample and study variables. As shown, means of MD-child and MD-adol were below the PERI clinical cut-off of 1.55 (Shrout, Dohrewend, & Levav, 1986). Fifteen percent of mothers were above the clinical cut-off at Time 1 and seven percent of mothers were above the clinical cut-off at Time 2. The skewness and kurtosis of all study variables, with the exception of cortisol, fell within the acceptable range (skewness cut-off - 2.0 and and kurtosis cut-off - 7.0; West, Finch, & Curren, 1995). The cortisol measures were log transformed to adjust for non-normality (Nicholson, 2007).

Table 2 shows zero-order correlations between the cortisol measures and all study variables. For these correlations, “cortisol reactivity” was calculated as the difference between P3 and P2 cortisol values. This definition of reactivity was selected because there was a general increase in cortisol levels from P2 to P3. There appeared to be minimal change in cortisol production from P1 to P2. The trapezoidal method was used to calculate “total cortisol” using area under the

curve with respect to ground (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003). Cortisol reactivity and total cortisol were calculated with raw data and then log transformed. MD-child was significantly negatively related to P2 cortisol, P3 cortisol, P4 cortisol, and total cortisol output. MD-adol was only marginally negatively correlated with P2 and P4 cortisol. Additionally, MD-child was significantly related to MD-adol ($r = .71$; $p < .01$) and MD-adol was significantly related to externalizing symptoms assessed in young adulthood ($r = .39$; $p < .05$).

*Identification of covariates:* The following demographic variables were examined as potential covariates: YA age, YA gender, baseline family income, ethnicity, use of oral or hormonally-based contraception, use of medications, smoking status, caffeine intake, and time of day when cortisol samples were taken (Kudielka, Hellhammer, & Wust, 2009). Psychological variables (i.e., Time 6 maternal depression, Time 6 internalizing symptoms, and Time 6 externalizing symptoms) were also examined (Kudielka, Hellhammer, & Wust, 2009). Zero order correlations between these variables and the six cortisol measures (i.e., four cortisol periods [P1,P2,P3, P4], cortisol reactivity, and total cortisol) were computed to identify variables that were significantly ($p < .05$) related to the cortisol measures (Table 2). Time of day was negatively related to P4 cortisol ($r = -.32$; $p < .05$), externalizing problems were negatively related to P3 ($r = -.36$; $p < .05$) and total cortisol ($r = -.30$; $p < .05$), and ethnicity was positively related to P4 cortisol ($r = .32$; $p < .05$), therefore these three variables were entered into statistical models as covariates.

*Regression diagnostics:* Regression diagnostics were conducted using separate regression equations with the four cortisol periods, cortisol reactivity, and total cortisol as dependent variables. Each regression equation contained the two predictor variables (MD-child and MD-adol) and three covariates (time of day, externalizing symptoms, and ethnicity). DFFITS, a measure of the influence of individual cases on the regression equation, and DFBETAS, a measure of the change in regression coefficients, were examined to identify potential outliers (Cohen, Cohen, West & Aiken, 2003). Cases were considered influential if the absolute value of DFFITS exceeded 1 or DFBETAS was greater than 1(Neter, Wasserman, & Kutner, 1989). No cases appeared to influence the regression of the predictors on the cortisol measures.

*Primary Analyses*

Repeated measures analysis of covariance (ANCOVA) was used to examine the effect of maternal depression on cortisol activity. MD-child, MD-adol, and the interaction between the two were regressed on the four repeated measures of cortisol (P1 – P4) controlling for time of day, externalizing symptoms, and ethnicity. MD-child significantly predicted lower overall cortisol output ($F = 7.64$; $p = .009$; see Table 3) and contributed to the prediction of cortisol at all four time points (P1: $\beta = -.386$, $p = .029$; P2: $\beta = -.465$ $p = .014$; P3: $\beta = -.507$, $p = .010$; P4: $\beta = -.466$, $p = .012$). Specifically, higher MD-child related to lower cortisol production at each time point. Additionally, the interaction between MD-child and MD-adol was marginally significant ($F = 3.03$; $p = .091$; partial $\eta^2 = .084$). The marginal interaction was plotted for additional

interpretation (Figure 1). Regression lines of MD-adol one standard deviation below the mean, at the mean, and one standard deviation above the mean were plotted as a function of MD-child and total cortisol output. The simple slopes of low ($b = -.410$; $p < .01$) and average ($b = -.303$; $p < .05$) MD-adol were significant, but the slope of high MD-adol was not significant ($b = -.196$; $p = .10$). Visual inspection of the graph revealed that high maternal depression at both time points contributed to the lowest cortisol levels and low cortisol at both time points contributed to the lowest cortisol levels. The interaction significantly contributed to the prediction of P2 cortisol ($\beta = .246$, $p = .041$) (Figure 2) and marginally contributed to the prediction of P1 cortisol ($\beta = -.192$, $p = .088$). There was a significant effect of externalizing symptoms on cortisol reactivity ($F = 3.48$; $p = .027$). Higher externalizing symptoms were related to lower cortisol reactivity (P3 – P2). There were no significant within-subject effects of maternal depression on cortisol reactivity (Table 3).

## DISCUSSION

The current study is unique in that it examines the relations between exposure to maternal depressive symptoms in childhood and adolescence and cortisol activity in young adulthood. Specifically, the study assessed the relations between depressive symptoms in divorced mothers and their offspring's cortisol reactivity and total cortisol output 15 years later. As predicted, higher levels of maternal depressive symptoms occurring in offspring's late childhood (9-12 years old) were significantly related to lower total cortisol output during a stress task.

The relation between maternal depressive symptoms occurring in late adolescence (15 – 19 years old) and total cortisol output in young adults was not significant. There was a marginal interaction between maternal depressive symptoms occurring in late childhood and depressive symptoms in adolescence, indicating that high maternal depressive symptoms at both time points was related to the lowest cortisol output. Neither depressive symptoms at either time point nor the interaction between the two was related to the magnitude of cortisol reactivity to the stress task.

Results of the current study are consistent with previous findings that maternal depression is related to dysregulated cortisol output in offspring (e.g., Ashman et al., 2002; Murray et al., 2009; Gump et al., 2009). Similar to previous investigations of offspring in childhood and adolescence, the current study found significant negative relations between maternal depressive symptoms and cortisol output in offspring, and non-significant relations between depressive symptoms and offspring's cortisol reactivity. This pattern of findings may further elucidate the nature of the effect of maternal depression on stress response and cortisol activity in offspring. Maternal depression is likely to be a more chronic, daily interpersonal environmental stressor rather than an acute, traumatic stressor (DeBellis, 2001). The physiological consequences in offspring, therefore, may manifest themselves in cortisol levels that are produced in the body throughout the day rather than those that are produced in response to an acute stressor. To respond to chronic stressors, such as maternal depression, it may be adaptive for the HPA-axis to down regulate the available levels of cortisol, so that elevated

amounts of cortisol are not produced throughout the day (Gunnar & Vasquez, 2001). This HPA-axis adaptation would not change the magnitude of cortisol reactivity in response to an acute stressor. Although some researchers have suggested that blunted cortisol levels may be an adaptive initial response to chronic stressors (Bevans, Cerbone, & Overstreet, 2008; Garmezy & Rutter, 1983), the persistence of reduced levels of cortisol over a long periods of time have been shown to have negative health-related consequences (McEwen, 2000; McEwen, 1998).

The significant finding that exposure to depression in late childhood is related to lower cortisol output in young adulthood is consistent with the emerging literature on hypocortisolism, where attenuated cortisol production is viewed as a long-term effect of exposure to early life stressors (Miller, Chen, & Zhou, 2007). Recently researchers have theorized that hypercortisol, or increased cortisol production, may be a short-term effect of environmental stressors and hypocortisol, or blunted cortisol production, may be a long-term result of environmental exposure to stress (Bevans et al., 2008; Tarullo & Gunnar, 2006). The lack of cortisol measures at earlier time points prevented examination of the pattern of change over time in the current sample, though it is possible that elevated cortisol activity might have been the more immediate response of children growing up with mothers having depressive symptoms, but, as more time passed, these increased cortisol levels may have triggered the HPA-axis in the offspring to instead produce lower amounts of cortisol.

In the context of the larger literature on maternal depression, the findings support the hypothesis that in older offspring, exposure to maternal depression may be related to blunted cortisol production. In general, studies of younger infants and children show that offspring of depressed mothers tend to exhibit higher basal cortisol levels than offspring of non-depressed mothers (Dougherty, Klein, Olino, Dyson, & Rose, 2009; Essex et al., 2002; Ashman et al., 2002; Murray et al., 2009). Studies examining offspring in late childhood and adolescence have been less consistent, revealing either attenuated cortisol output in offspring of mothers with high levels of depression relative to offspring of mothers with lower levels of depression (Gump et al., 2009) or a non-significant relation between maternal depression and cortisol reactivity in offspring (Ronsaville et al., 2006). The current study extends the literature by its assessment of the relation between maternal depressive symptoms and cortisol activity in young adult offspring. Findings suggest that the blunted cortisol levels that have been observed in offspring in late childhood and adolescence are maintained into the next developmental stage.

Prior to this investigation, the study of maternal depression and HPA axis activity in offspring was focused on maternal depression occurring in the offspring's infancy and early childhood. There appears to be a sensitive period in the prenatal, infant and toddler stages when children are particularly susceptible to the effects of depression in their mothers (e.g., Dougherty, Klein, Olino, Dyson, & Rose, 2009; Essex et al., 2002). The current findings suggest there may be an additional sensitive period in late childhood when maternal depressive

symptomotology continues to affect cortisol activity in offspring. Interventions aimed at ameliorating depression in mothers should focus both on mothers with infants as well as mothers with children in this late childhood/ early adolescent stage. Future studies should compare the mechanisms that may explain the harmful effects of maternal depression during these two sensitive periods. Pinpointing these mechanisms will help in the development of interventions that may benefit both mothers and their children in the short- and long-term.

It is interesting to speculate about why maternal depressive symptoms occurring in late childhood significantly related to cortisol activity in young adult offspring. Additionally, it is important to consider what is unique about this stage that may make children susceptible to their mother's depressive symptoms. Late childhood/early adolescence is a period characterized by many stressful changes, such as beginning pubertal development, school transition, and navigating new relationships (Simmons, Burgeson, & Carlton-Ford, 1987). These stressors occur when youths' brains are still developing, which may make it more difficult to regulate emotion and cope with internal and external stressors (Dahl, 2004). Supportive relationships can help children in this developmental stage handle these stressors, and may decrease psychological distress in offspring (DuBois, Felner, Brand, Adan, & Evans, 2002). Depressive symptoms may decrease the support mothers are able to provide for their children. For example, maternal depression is linked with changes in parenting like decreased warmth, increased negativity, and disengagement from their child (Goodman & Gotlib, 1999; Lovejoy et al., 2000). Children in late childhood/early adolescence may be more

susceptible than older adolescents to the negative effects these suboptimal parenting behaviors can have on the stress response system (DeBellis, 2001; Bugental, Martorell, & Barraza, 2003), and these consequences may persist into young adulthood. Alternatively, mothers with depression may model maladaptive coping strategies such as hostility and ineffective problem solving (Blandon et al., 2008; Goodman & Gotlib, 2002; Gump et al., 2009). Their children may incorporate the use of these strategies into their repertoire of dealing with stressors, which may lead to a dysregulated cortisol activity (Repetti et al., 2002).

Although not central to the aims of the study, the findings revealed that current externalizing symptoms were significantly related to cortisol reactivity, with higher levels of externalizing symptoms related to lower reactivity. This effect of externalizing symptoms was separate from and did not account for the main effect of maternal depressive symptoms in late childhood on total cortisol output in offspring. This result is consistent with the emerging hypothesis that externalizing symptoms are related to hypocortisol responses in young adults (e.g., Lahey, McBurnett, Loeber, & Hart, 1995; Van Goozen, 2005; Van Goozen, Fairchild, Snoek, & Harold, 2007). However, findings in support of this theory have been mixed. A meta-analysis conducted by Alink and colleagues (2008) found that externalizing was significantly related to lower cortisol levels in school-age children, but the relation between externalizing problems and cortisol activity in adolescents was non-significant. More recently, a study of parentally-bereaved young adults revealed a negative association between externalizing symptoms and cortisol output during a stress task (Luecken et al., 2010). It may

be that the nature of the association between externalizing problems and cortisol levels changes across development. Additional studies are needed to explore the relation between externalizing symptoms and cortisol activity across developmental stages.

There are several limitations to the current study. First, the sample size is small, which may have contributed to a lack of power to adequately test the interaction between exposure to maternal depressive symptoms in late childhood and exposure in adolescence. Second, cortisol was measured only during young adulthood, limiting the ability to examine whether the nature of the relation between cortisol and maternal depressive symptoms changes over the course of development. Third, given the correlational design of this study, causal inferences cannot be made. The observed significant relation may be due to other variables associated with maternal depression, such as exposure to heightened levels of other environmental stressors. Fourth, the measure used to assess maternal depressive symptoms (i.e., PERI) was not a direct measure of depression, but rather a measure of general demoralization. Although this measure has been shown to be a good predictor of depressive symptomotology (Roberts & Vernon, 1981; Tanaka & Huba, 1984), it is not based on DSM-IV criteria for clinical depression. Additionally, few mothers exceeded the PERI clinical cut-off levels for demoralization and although the current study examined divorced women, a group at higher risk for depression (Lorenz, Wickrama, Conger, & Elder, 2006), the depression levels in the sample were consistent with levels in the general population. Fifth, given that the data on maternal depressive symptoms were only

collected at baseline, six months later, six years later and 15 years later, the effect of chronic maternal depression on cortisol activity in young adult offspring could not be directly assessed. It is possible that the effect attributed to maternal depressive symptoms occurring in late childhood was a carryover effect of depressive symptoms occurring earlier in development. Additional studies are needed to determine whether late childhood is a distinct sensitive period in which children are susceptible to the effects of depressive symptoms in their mothers.

Future studies should recruit larger samples that would provide the power to examine more adequately interactions between maternal depressive symptoms occurring at different time periods to explore the relation between chronic maternal depression and cortisol activity in offspring. Future investigations that are longitudinal in design and assess cortisol and depression measures at many time points would allow the examination of changes in the relations between maternal depression and offspring's cortisol activity over time. Future studies should also employ more direct measures of depression based on DSM-IV criteria (e.g. Diagnostic Interview Schedule for the DSM-IV (DIS-IV); Robins et al., 2000). Similarly, samples should include mothers with clinical levels of depression in order to better assess the influence of depressive symptoms on cortisol activity in offspring. Finally, future studies should examine the mechanisms, such as changes in parenting behaviors, through which maternal depression negatively influences cortisol regulation.

The current study extends the existing literature by examining the prospective relations between exposure to maternal depressive symptoms in late

childhood and adolescence and cortisol activity in young adult offspring. Findings showed that maternal depressive symptoms occurring in late childhood, but not in adolescence, were related to blunted total cortisol output. This attenuated cortisol production may put these young adult offspring at increased risk for a variety of adverse physical and mental health consequences (McEwen, 2000; McEwen, 1998). Additionally, results point to a potential sensitive period when maternal depression may have a negative effect on future functioning in their children that is worthy of further investigation. Increasing the understanding of the mechanisms by which maternal depression can affect cortisol activity in offspring may have longer-term benefits for both mental and physical health in offspring than previously believed.

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Table 1. Descriptive information on study variables

| Variable | Mean (SD) | Range | Skewness | Kurtosis |
|---|---|---|---|---|
| MD-child | 1.12 (.50) | .46 – 2.93 | 1.31 | 2.82 |
| MD-adol | .796 (.48) | .30 – 2.59 | 1.74 | 3.83 |
| Current maternal depression | .640 (.53) | .00 – 2.56 | 1.88 | 3.98 |
| Internalizing symptoms | 54.2 (4.2) | 50.0 – 65.5 | .861 | .014 |
| Externalizing symptoms | 48.8 (8.2) | 32.5 – 60.5 | -.386 | -1.16 |
| Cortisol P1 (ug/dl) | .098 (.06) | .023 - .350 | 2.00 | 6.50 |
| Cortisol P2 (ug/dl) | .095 (.06) | .012 - .260 | 1.15 | 1.57 |
| Cortisol P3 (ug/dl) | .103 (.07) | .018 - .332 | 1.26 | 1.18 |
| Cortisol P4 (ug/dl) | .083 (.05) | .013 - .240 | 1.22 | 1.15 |
| Reactivity (ug/dl) | .012 (.15) | -.46 - .29 | -.514 | 1.31 |
| Total Output (ug/dl) | .431 (.26) | .07 – 1.30 | 1.21 | 1.84 |

Table 2. Zero-order Correlations with Cortisol

| | Period 1 | Period 2 | Period 3 | Period 4 | Reactivity | Total |
|---|---|---|---|---|---|---|
| Time of Day | .24 | .22 | -.25 | **-.32*** | -.10 | **-.28†** |
| BMI | .06 | -.08 | -.12 | -.14 | -.10 | -.09 |
| Age | -.03 | .11 | .04 | .07 | .09 | .09 |
| Gender | .02 | -.12 | -.21 | -.13 | -.21 | .02 |
| Ethnicity | .22 | .14 | .17 | **.32*** | .10 | .20 |
| Oral contraceptive | .02 | -.32 | -.19 | -.35 | .20 | -.30 |
| Exercise | .06 | .12 | .09 | .16 | .01 | .10 |
| Smoking (Y/N) | -.09 | .04 | .05 | .07 | .11 | .06 |
| Internalizing | .01 | .02 | -.10 | -.05 | -.24 | -.01 |
| Externalizing | -.16 | -.26 | **-.36*** | -.21 | -.24 | **-.31*** |
| MD-child | **-.30†** | **-.35*** | **-.40*** | **-.48**** | -.17 | **-.30*** |
| MD-adol | -.17 | **-.27†** | -.22 | **-.29†** | .06 | -.18 |
| T6 Maternal depression | -.03 | -.13 | -.20 | -.24 | -.18 | -.16 |

**p < .01; *p < .05; †p < .10

Note: Cortisol measures are log-transformed. Gender is coded as 1 = male, 2 = female. Ethnicity is coded as Caucasian = 1, other = 0.

Table 3. Summary of Repeated Measures Analysis of Covariance

| **Total Output** | **df** | **F** | ***p*** | **partial $\eta^2$** |
|---|---|---|---|---|
| MD-child | 1,33 | **7.64** | **.009** | .188 |
| MD-adol | 1,33 | .784 | .382 | .023 |
| MD-child*MD-adol | 1,33 | **3.03** | **.091** | .084 |
| Time of Day | 1,33 | .361 | .552 | .011 |
| Externalizing Symptoms | 1,33 | 1.82 | .186 | .052 |
| Ethnicity | 1,33 | .077 | .783 | .002 |
| **Reactivity** | **df** | **F** | ***p*** | **partial $\eta^2$** |
| Period | 3, 80 | .435 | .688 | .013 |
| Period * MD-child | 3, 80 | .476 | .661 | .014 |
| Period * MD-adol | 3, 80 | 2.14 | .114 | .061 |
| Period * MD-child*MD-adol | 3, 80 | .862 | .445 | .025 |
| Period * Time of Day | 3, 80 | .284 | .796 | .009 |
| Period * Externalizing Symptoms | 3, 80 | **3.48** | **.027** | .095 |
| Period * Ethnicity | 3, 80 | 1.28 | .283 | .038 |

Figure 1. Total Cortisol Output Interaction

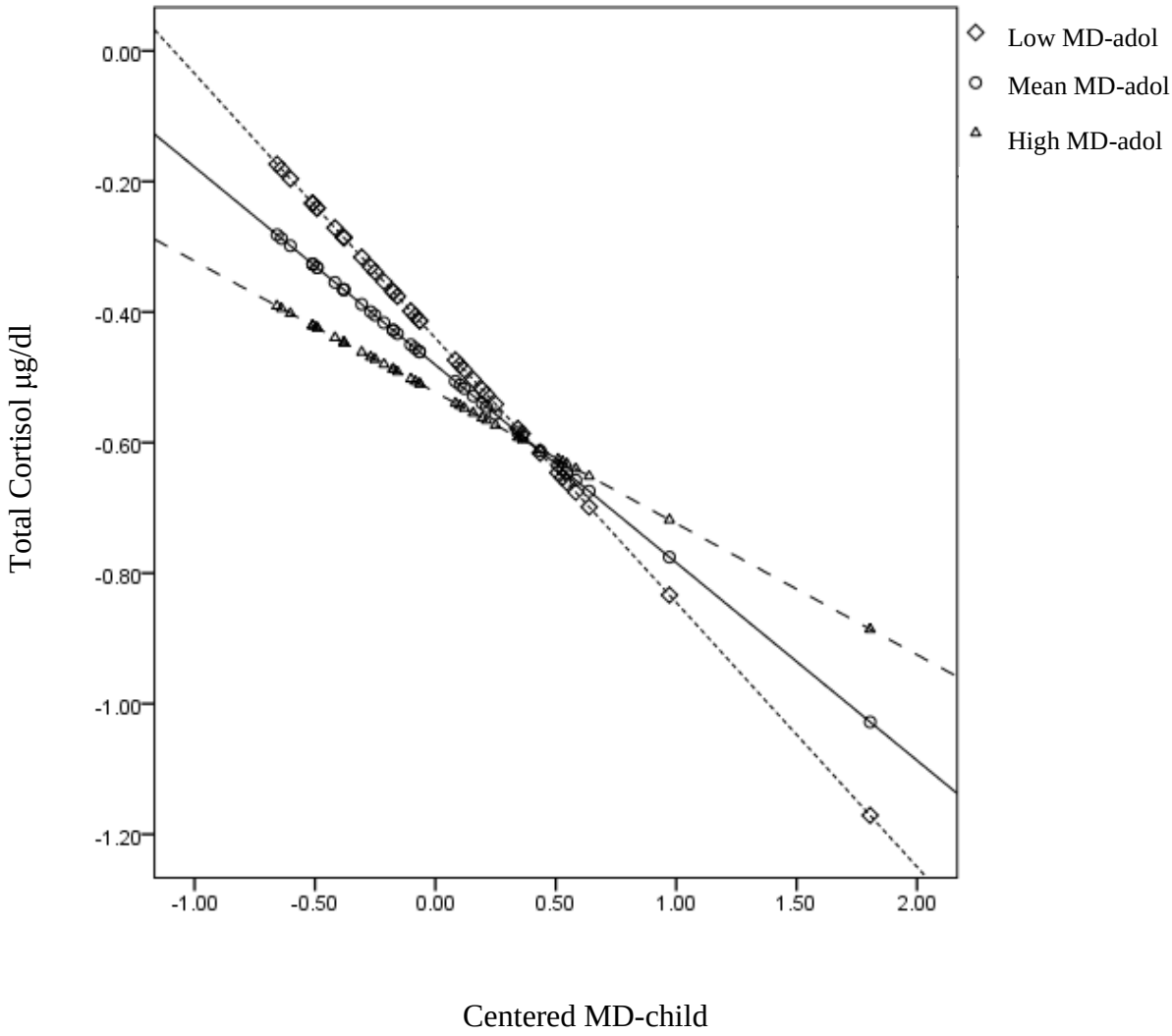
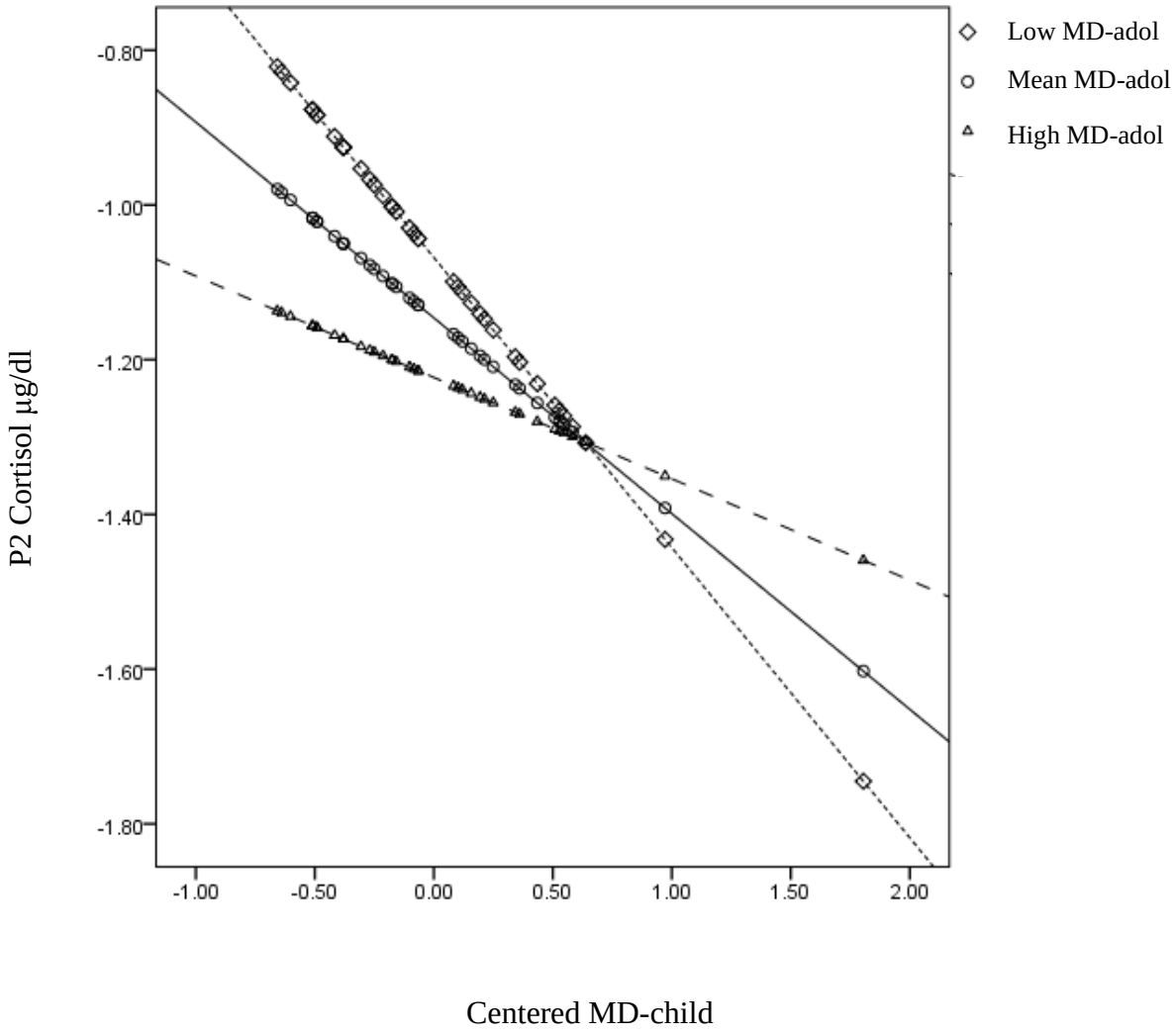


Figure 2. P2 Cortisol Interaction



# Appendix A

## PSYCHIATRIC EPIDEMIOLOGY RESEARCH INTERVIEW

## (PERI)

*Mother Report*

Directions

In this set of questions, we'd like to know more about YOU in the PAST MONTH.

| | |
|---|---|
| 1. | In the past month, how often have you felt bothered by all different kinds of ailments in different parts of your body? |
| 2. | How often have you been bothered by feelings of sadness or depression -- feeling blue? |
| 3. | How often have you had attacks of sudden fear or panic? |
| 4. | How often have you felt confident? |
| 5. | How often have you felt lonely? |
| 6. | How often have you been bothered by feelings of restlessness? |
| 7. | How often have you felt useless? |
| 8. | How often have you feared going crazy; losing your mind? |
| 9. | Remember, we are talking about how you have felt during the past month. How often have you felt anxious? |
| 10. | How often have you acted like a coward? |
| 11. | How often have you feared something terrible would happen to you? |
| 12. | How often have you felt confused and had trouble thinking? |
| 13. | How often have you had trouble concentrating or keeping your mind on what you were doing? |
| 14. | How often have you done anything of a sexual nature that society does not approve of? |
| 15. | How often have you felt that nothing turns out for you the way you want it to? |
| 16. | How often have you felt completely hopeless about everything? |
| 17. | How often have you felt completely helpless? |
| 18. | Please stay focused on the past month. How often have you had times when you couldn't help wondering if anything was worthwhile anymore? |
| 19. | How often have you been bothered by cold sweats? |
| 20. | How often have you had trouble with headaches or pains in the head? |
| 21. | How often do you eat too much? |
| 22. | How often has your appetite been poor? 23. |

| | |
|---|---|
| 23. | When you have gotten angry, how often have you felt uncomfortable, like getting headaches, stomach pains, cold sweats and things like that? |
| 24. | If it is more convenient for you to do so, how often will you tell a lie? |
| 25. | You are the kind of person who feels (he/she) has much to be proud of. |
| 26. | You are the kind of person who is the worrying type, you know, a worrier. |
| 27. | You are the kind of person who feels that (he/she) is a failure generally in life. |

APPENDIX B

# BRIEF HEALTH SURVEY

*Young Adult Report*

Directions

The next questions concern your health-related behaviors. Please remember that all of your answers will be kept confidential. Please answer all questions honestly and completely.

| | |
|---|---|
| 1a | Please estimate how many servings of caffeinated beverages such as COFFEE, TEA, COLA or MOUNTAIN DEW do you drink in an average day (one serving = one cup, bottle, or glass) |
| 1b | How many servings have you had so far today? |
| 2a | Please estimate how many servings of energy drinks that you consume in an average day? These are drinks like Red Bull or Throttle. (one serving = one cup, bottle, or glass) |
| 2b | How many servings have you had so far today? |
| 3a | Do you currently smoke cigarettes or cigars? IF NO, GO ON TO QUESTION 4 |
| 3b | How many do you smoke in an average day? |
| 3c | Approximately what time did you last smoke today? |
| 4 | How tall are you? |
| 5 | How much do you weigh? |
| 6 | On average, how many servings of alcohol do you consume in a week? (one serving= 1 beer, shot of liquor, or glass of wine) |
| 7a | Do you ever drink alcohol? |
| 7b | How much did you drink at that time? (one serving= 1 beer, shot of liquor, or glass of wine) |
| 7c | How much did you drink at that time? (one serving=1 beer, shot of liquor, or glass of wine) |
| 8a | Did you exercise today? IF NO, GO TO QUESTION 9a |
| 8b | What time did you exercise? |
| 8c | How long did you exercise (in minutes)? |
| 9a | Did you eat before coming here today? IF NO, GO TO QUESTION 10a |
| 9b | What time did you eat? |

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| 9c | What did you eat? |
| 10a | Are you currently taking any medication? |
| 10b | How many medications are you currently taking? |
| 10c | Please list the first/next medication you are taking. |
| 10d | Please give the reasons for taking the medication. |
| 10e | When was the last time you took the medication? |
| 11a | Do you currently have any medical conditions for which you have received or are receiving care from a physician? |
| 11b | How many current medical conditions are there for which you have received or are receiving care from a physician? |
| 11c | Please list any current medical conditions for which you have received or are receiving care from a physician. |
| 12 | Do you take or receive oral or hormonally-based contraceptives? |
| 13 | Approximate date last menstrual period began |
| 14 | How old were you when you began menstruating? |

APPENDIX C

# ADULT SELF-REPORT (ASR)/ ADULT BEHAVIOR CHECKLIST (ABC)

*Young Adult Report and Mother Report*

Directions

*Young Adult*

Next I will read a list of items that describe people. As I read each item, please decide whether it has been true of you over the past 6 months, since [6-month marker]. Please answer all items as well as you can, even if some do not seem to apply to you.

*Mother*

Next I will read a list of items that describe people. As I read each item, please decide whether it has been true of [YA name] over the past 6 months. Please answer all items as well as you can, even if some do not seem to apply to [YA name]

*Note: ASR questions are in first person. ABC questions are equivalent but in third person.*

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| 1 | I am too forgetful |
| 2 | I make good use of my opportunities |
| 3 | I argue a lot |
| 4 | I work up to my ability |
| 5 | I blame others for my problems |
| 6 | I use drugs (other than alcohol or nicotine) for nonmedical purposes |
| 7 | I brag |
| 8 | I have trouble concentrating or paying attention for long |
| 9 | I can't get my mind off certain thoughts |
| 10 | I have trouble sitting still |
| | *Remember, the following items refer to whether these have been true of you over the past 6 months.* |
| 11 | I am too dependent on others |
| 12 | I feel lonely |
| 13 | I feel confused or in a fog |
| 14 | I cry a lot |
| 15 | I am pretty honest |
| 16 | I am mean to others |
| 17 | I daydream a lot |
| 18 | I deliberately try to hurt or kill myself |
| 19 | I try to get a lot of attention |
| 20 | I damage or destroy my things |
| 21 | I damage or destroy things belonging to others |
| | *Keep thinking of whether these have been true of you over the past 6 months..* |
| 22 | I worry about my future |
| 23 | I break rules at work or elsewhere |
| 24 | I don't eat as well as I should |
| 25 | I don't get along with other people |
| 26 | I don't feel guilty after doing something I shouldn't |
| 27 | I am jealous of others |
| 28 | I get along badly with my family |

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| 29 | I am afraid of certain animals, situations, or places |
| 30 | My relations with the opposite sex are poor |
| 31 | I am afraid I might think or do something bad |
| 32 | I feel that I have to be perfect |
| | *Keep thinking of whether these have been true of you over the past six months.* |
| 33 | I feel that no one loves me |
| 34 | I feel that others are out to get me |
| 35 | I feel worthless or inferior |
| 36 | I accidentally get hurt a lot |
| 37 | I get in many fights |
| 38 | My relations with neighbors are poor |
| 39 | I hang around people who get in trouble |
| 40 | I hear sounds or voices that other people think aren't there |
| 41 | I am impulsive or act without thinking |
| 42 | I would rather be alone than with others |
| 43 | I lie or cheat |
| | *Keep thinking of whether these have been true of you over the past six months.* |
| 44 | I feel overwhelmed by my responsibilities |
| 45 | I am nervous or tense |
| 46 | Parts of my body twitch or make nervous movements |
| 47 | I lack self-confidence |
| 48 | I am not liked by others |
| 49 | I can do certain things better than other people |
| 50 | I am too fearful or anxious |
| 51 | I feel dizzy or lightheaded |
| 52 | I feel too guilty |
| 53 | I have trouble planning for the future |
| 54 | I feel tired without good reason |
| 55 | My moods swing between elation and depression |
| | *The next few questions are about physical problems without known medical cause.* |
| 56a | Aches or pains (not stomach or headaches) |
| 56b | Headaches |
| 56c | Nausea, feels sick |
| 56d | Problems with eyes (not if corrected by glasses) |
| 56e | Rashes or other skin problems |
| 56f | Stomachaches |
| 56g | Vomiting, throwing up |
| | *That ends the section on physical problems without known medical cause. Just continue using your same answer list for the next set of questions. Keep thinking of whether it has been true of you over the past 6 months.* |
| 57 | I physically attack other people |
| 58 | I pick my skin or other parts of my body |
| 59 | I fail to finish things I should do |
| 60 | There is very little that I enjoy |
| 61 | My work performance is poor |
| 62 | I am poorly coordinated or clumsy |
| 63 | I would rather be with older people than with people of my own age |
| 64 | I have trouble setting priorities |
| 65 | I refuse to talk |
| 66 | I repeat certain acts over and over |
| 67 | I have trouble making or keeping friends |

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| | *Keep thinking of whether these have been true of you over the past 6 months..* |
| 68 | I scream or yell a lot |
| 69 | I am secretive or keep things to myself |
| 70 | I see things that other people think aren't there |
| 71 | I am self-conscious or easily embarrassed |
| 72 | I worry about my family |
| 73 | I meet responsibilities to my family |
| 74 | I show off or clown |
| 75 | I am too shy or timid |
| 76 | My behavior is irresponsible |
| 77 | I sleep more than most other people during day and/or night |
| 78 | I have trouble making decisions |
| 79 | I have a speech problem |
| | *Keep thinking of whether these have been true of you over the past six months.* |
| 80 | I stand up for my rights |
| 81 | My behavior is very changeable |
| 82 | I steal |
| 83 | I am easily bored |
| 84 | I do things that other people think are strange |
| 85 | I have thoughts that other people would think are strange |
| 86 | I am stubborn, sullen, or irritable |
| 87 | My moods or feelings change suddenly |
| 88 | I enjoy being with people |
| 89 | I rush into things without considering the risks |
| 90 | I drink too much alcohol or get drunk |
| | *Keep thinking of whether these have been true of you over the past six months.* |
| 91 | I think about killing myself |
| 92 | I do things that may cause me trouble with the law |
| 93 | I talk too much |
| 94 | I tease others a lot |
| 95 | I have a hot temper |
| 96 | I think about sex too much |
| 97 | I threaten to hurt other people |
| 98 | I like to help others |
| 99 | I dislike staying in one place for very long |
| | *Keep thinking of whether these have been true of you over the past six months.* |
| 100 | I have trouble sleeping |
| 101 | I stay away from my job even when I'm not sick and not on vacation |
| 102 | I don't have much energy |
| 103 | I am unhappy, sad, or depressed |
| 104 | I am louder than others |
| 105 | People think I am disorganized |
| 106 | I try to be fair to others |
| 107 | I feel that I can't succeed |
| 108 | I tend to lose things |
| 109 | I like to try new things |
| | *Keep thinking of whether these have been true of you over the past six months.* |
| 110 | I wish I were of the opposite sex |
| 111 | I keep from getting involved with others |
| 112 | I worry a lot |
| 113 | I worry about my relations with the opposite sex |

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| 114 | I fail to pay my debts or meet other financial responsibilities |
| 115 | I feel restless or fidgety |
| 116 | I get upset too easily |
| 117 | I have trouble managing money or credit cards |
| 118 | I am too impatient |
| 119 | I am not good at details |
| 120 | I drive too fast |
| 121 | I tend to be late for appointments |
| 122 | I have trouble keeping a job |
| 123 | I am a happy person |
| | *For the next few questions, you don't need to use your list. Just tell me what you think. Think of the past 6 months, from [6-month marker] up until now.* |
| 124 | In the past 6 months, about how many times per day did you use tobacco (including smokeless tobacco)? |
| 125 | In the past 6 months, on how many days were you drunk? |
| 126 | In the past 6 months, on how many days did you use drugs for nonmedical purposes (including marijuana, cocaine, and other drugs, except alcohol and nicotine)? |
| | *Use List 10 again to respond to the following statements.* |
| 127 | I act too young for my age |
| 128 | I break rules at school or work |
| 129 | I get teased a lot |
| 130 | I bite my fingernails |
| 131 | I eat too much |
| 132 | I am overweight |
| 133 | I would rather be with younger people than with people my own age |
| 134 | I set fires |
| 135 | I store up things I don't need |
| 136 | I am suspicious |
| 137 | I like to take life easy |